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ABSTRACT

Two groups of 10 children between the ages of 94 and 165 months were paired for age (within 6 months) and sex and were compared for Brainstem Auditory Evoked Response patterns. One child in each pair showed evidence of central language disturbance as determined by neuropsychological testing. The other did not. All had normal hearing and IQs of 80 or above. Ss with central language disturbance showed longer latencies and interwave transmission times than did controls. The segments in which the differences reached significance did not extend past wave III. Findings indicated that Ss with central language disturbance may have auditory stimuli transmission disturbances below the level of the olivary nuclei. It is speculated that at least a portion of the slowed brainstem transmission findings in many children with diagnoses of autism, retardation, and minimal brain dysfunction may relate to language disturbance. (CL)

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Brainstem Auditory Evoked Response in Children with Central Language Disturbance

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Two groups of 10 children paired for age and sex were compared for Brainstem Auditory Evoked Response (BSAER) patterns. One child in each pair showed evidence of central language disturbance as determined by neuropsychological testing. The other did not. All had normal hearing and IQs of 80 or above. The children with central language disturbance showed longer latencies and interwave transmission times than did the controls. The segments in which the differences reached significance did not extend past wave III. The findings indicate that children with central language disturbance may have auditory stimuli transmission disturbances below the level of the olivary nuclei.

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Bilateral surgical lesions of the inferior colliculi of cats resulted in a marked decrease in their response to sound, even though their hearing was intact (Jane et al., 1965). They found that normal cats are more likely to respond to sound than to light when the two stimuli are presented simultaneously. Bilateral lesions of the auditory cortex and of the trapezoid body did not change this hierarchy. Lesions of the inferior colliculi reversed it. The animals were then less responsive to sound than to light stimuli. Jane et al. (1965) also demonstrated that lesions confined to the inferior colliculi resulted in no apparent sensory deficit in sound localization tasks, while lesions extending into the brachium disrupted left ear vs. right ear discrimination, and also eliminated auditory evoked potentials recorded at the level of the auditory cortex. Denny-Brown (1962) had previously reported that surgical lesions involving both the superior and inferior colliculi in monkeys resulted in animals that seemed to be unaware of their environment and who spent their time staring into space, although they would explore for food when hungry and showed a normal sleep pattern.

Simon (1975) hypothesized that lesions of the brainstem auditory nuclei could, without causing deafness, interfere with a child's ability to discriminate various features of speech and therefore disturb his or her language development. In 1978, Student and Sohmer reported on Brainstem Auditory Evoked Response (BSAER) studies on 15 children with early onset autistic traits as compared to normal control children. Five of the autistic children showed profound cochlear hearing loss. The other 10 autistic children as a group showed significantly longer latencies for waves I through waves V than did the control children. They stated that their findings were indicative of the presence of organic disturbance in the brainstem pathways—possibly due to slowed development or immaturity of brainstem mechanisms. Student and Sohmer (1979) later reported that there had been a malfunction in their equipment. On reanalysis of their data they found that though the latencies were still longer in the autistic children, the differences did not reach significance. Rosenblum et al. (1980), in a study of six autistic children pair matched for age and sex to normal children, found that the latencies for waves III and IV and the transmission time, as measured from the positive peak of wave I to the negative peak of wave IV, were significantly longer in the autistic children. Another group, (Fein et al., 1981) compared the BSAER in a group of 16 autistic children and a group of 20 normal control children. They found that the interpeak I–V latency was significantly longer bilaterally and the interpeak III–V latency was significantly longer on stimulation of the left ear for the autistic as compared to the normal controls. They also subdivided the autistic children into two groups: those who showed BSAER latency abnormalities and those who did not. They compared the behaviors of these two subgroups of autistic children and found that symptoms primarily involving social aloofness and an abnormally long attention span for certain activities, i.e., perseverative play, were significantly greater in the subgroup of autistic children showing the brainstem latency abnormalities.

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Salamy and Mendelson (1980) studied 245 normal full-term and 97 infants who had sustained various complications of pregnancy or birth. Many were premature. They measured and compared the BSAER of the two groups on 2–3 occasions between birth and 1 year of age. The latency of waves I and V were sufficiently longer in the high-risk infants in that these infants could be distinguished from the normal full-term infants during the first 3–5 months of life. After that the differences between waves I and V latencies was insufficient to distinguish the two groups. Wave III latencies began to diverge for the two groups during the latter part of the first year. The amplitudes of waves I, III, and V got progressively higher over time in the normal subjects, but tended to remain low in the high-risk infants. They state that the incidence of children with generalized cognitive impairments and neurological soft signs who present histories of unfavorable perinatal events is formidable. They speculated that focal brainstem and thalamic lesions may account for a variety of childhood disorders including infantile autism and minimal brain dysfunction. Cantwell et al. (1980) reported that 53 of 100 children seen consecutively at a suburban speech and hearing clinic were found to have psychiatric illness. They believe this indicates that children with speech and language disorders are at risk for the development of psychiatric disturbance.

Almost all children admitted, and some evaluated as outpatients, at Lafayette Clinic for psychiatric disturbance receive a battery of psychological and neuropsychological tests. This includes tests which are believed to reflect central language function. We hypothesized that those psychiatrically disturbed children who showed deficits on tests of central language function would show disturbances on their BSAER as compared to a group of children without evidence of language dysfunction.

Method

Two groups of 10 children paired for age (within 6 months) and sex were tested (mean age both groups 131 months, range 94–165 months). All children were medication free for at least 2 weeks prior to testing. All the experimental subject children were psychiatric patients. The control children consisted of psychiatric patients and normal children. Diagnoses included adjustment reaction, conduct disturbance, Tourette's syndrome, and attention deficit disorder. Their hearing was within normal limits as measured by audiometric screening. The screening consisted of pure tones at 7 frequencies (125, 250, 5000, 1000, 2000, 4000, and 8000 Hz) as recommended by the manufacturer (Maico). Twenty-five-decibel loss was used as the minimum threshold screening level. The audiometric screening was performed on the experimental subjects as part of the clinical neuropsychological evaluation. This preceded the BSAER testing (X = 7.8 months, median 7 months, range 1–21 months). The audiometric testing in seven of the controls was done the same day as the BSAER testing. In the other three it preceded the BSAER by 12, 11, and 1 month(s). The subject and control children received a Peabody Picture Vocabulary Test (PPVT). All the experimental subject children had received a WISC-R. All scored 80 or above on the Full Scale ($FS = 87$, $V = 85$, $P = 91$). None of the children were autistic.

The subject children were chosen on the basis of their scores on four neuropsychological tests: (a) Word Fluency (Benton, 1973), (b) Auditory Closure (Kass, 1964; Knights, 1966), (c) Speech-Sounds Perception (Reitan, 1969), and (d) Sentence Memory (Benton, 1965). They had to show a deficit of at least 2 standard deviations below the mean on either word fluency or auditory closure and at least 1 standard deviation below the mean on one or more of the remaining three tests. Word fluency and auditory closure were emphasized because we believed they involved the phonologic, morphologic, and semantic aspects of language as opposed to speech sounds perception and sentence memory, which we believed primarily involved semantics and short-term auditory memory, respectively. The majority of these children were receiving help for their language disturbance from our language pathologist. The control children had to show no deficits on any of the four tests. Informed parental permission and child assent was obtained.

Recording was performed by use of a Nicolet click generator, a Nicolet averaging computer and a Grass EEG machine. Leads used were the central, bilateral mastoid, and a forehead ground. These were attached to the scalp by collodion. Masking earphones were placed over the child's ears. Clicks were generated by alternating pulse polarity at the rate of 11.1/sec at 95 db sound level (SL). The electrical activity was amplified to a gain of 10^6 and filtered with a band pass of 150–3000 Hz. Each trial consisted of 2048 clicks. There were at least two trials for each ear. The clicks were delivered to each ear separately. The responses were recorded from leads at the vertex and mastoid ipsilateral to the stimulated ear. Measurements were made at the maximum positive peak for each wave. The latency and amplitude of each wave were measured; the results from replicated trials were averaged together to obtain a mean value for each ear. This mean was used for data analysis. The peaks were identified by an electroencephalographer. Ten of the tracings were chosen by use of a random numbers

table. These 10 tracings were read independently by two readers who identified the waves and then measured their latencies and amplitudes. Correlations did not reach significance for latency of waves VI and VII bilaterally and for wave IV (right ear stimulation). Correlations for waves I (left ear stimulation), wave II, III, and V bilaterally all exceeded 0.9. Correlation for wave I (right ear stimulation) was 0.636 which exceeded the 0.05 level of significance. Correlations for amplitudes of waves I, II, III, and IV–V all exceeded 0.9. Amplitude measurements were from the positive peak to the following negative peak for that wave. In amplitude measurements wave V actually refers to the IV–V complex.

Data Analysis

The data were analyzed by three factor analyses of variance (ANOVA) with two repeated measures. This was performed for the latency periods of waves I, II, III, and V but not for waves IV, VI or VII because of the amount of missing data for these three waves. It was also initially performed for the interwave transmission times I–III, I–V, and III–V. Because of the positive finding in the I–III interwave transmission time, the ANOVA was also done on the II–III and the I–II interwave transmission times. There were missing data for wave II on left ear stimulation in one experimental subject and on right ear stimulation in one other experimental subject. For purposes of the ANOVA analysis, the respective means for the other nine subjects were inserted for the two missing measurements. No data were missing for waves I, III or V. Two-tailed *t*-tests were done on the original measurements for waves I, II, III, IV, V, VI, and VIII rather than on the ANOVA means because of the missing data.

Results

The PPVT scores of the control group ($\bar{X} = 119$) were significantly higher than the experimental subject group ($\bar{X} = 92$), ($F = 19.838$, $df = 1,19$; $p < 0.01$). The ANOVA showed significant differences between the experimental subjects and the controls in the latencies and interwave transmission times for the first three waves (Table 1), but there were no significant latency or interwave transmission time differences for waves V or for the I–V and III–V interwave transmission times. The interaction between subject groups and laterality for the I–III interwave transmission time reached significance ($F = 8.000$; $df = 1,39$; $p < 0.05$). *t*-Tests showed the significant difference was on left ear stimulation where the I–III interwave transmission time was significantly longer for the experimental subjects than for the controls (2.18 ms vs. 2.04 ms), ($t = 2.264$; $df = 1,9$; $p < 0.05$). *t*-Tests showed no significant latency differences for waves IV, VI or VII. The ANOVA showed a significant main effect between subject groups for the II–III interwave transmission time, the experimental subjects showing a significantly longer II–III interwave transmission time than the controls ($F = 1.8151$; $df = 1,39$; $p < 0.05$). *t*-Tests only substantiated this difference as significant when the right ear was stimulated (1.03 ms vs. 0.93 ms) ($t = 3.000$, $df = 1,8$; $p < 0.05$). The I–II interwave transmission time controls vs. subjects showed no significant differences. *t*-Tests also showed significantly longer latencies for the experimental subjects than for the controls for wave III on left ear

TABLE 1

Latency and Interwave Durations (ms) Experimental Subjects (ES) vs. Controls (C)[a]

	Latency								Interwave Transmission Time									
	Wave I		Wave II		Wave III		Wave V		I–II		II–III		I–III		III–V		I–V	
	C	ES	C	ES	C	ES	C	ES	C	ES	C	ES	C	ES	C	ES	C	ES
Left																		
X	1.36	1.42	2.41	2.54	3.40	3.61[b]	5.30	5.44	1.04	1.12	1.00	1.06	2.04	2.18[c]	1.90	1.83	3.94	4.02
S.D.	0.09	0.15	0.13	0.15	0.15	0.14	0.20	0.17	0.10	0.08	0.11	0.11	0.13	0.17	0.18	0.10	0.20	0.17
Right																		
X	1.33	1.43[d]	2.48	2.50	3.43	3.58	5.34	5.43	1.16	1.09	0.93	1.03[e]	2.10	2.15	1.91	1.85	4.01	4.00
S.D.	0.09	0.08	0.13	0.08	0.16	0.19	0.23	0.22	0.12	0.09	0.08	0.07	0.14	0.16	0.24	0.10	0.25	0.20

[a] I–III interwave duration on right side for experimental subjects of 2.15 ms is longer than the sum of its two segments, I–II and II–III, which equal 2.12 ms. This due to missing data for wave II on one of the experimental subjects so the II–III and I–II interwave durations were calculated with an *N* of 9 and the I–III interwave duration was calculated with an *N* of 10. This is also the reason for the discrepancy found if using the wave I, II and III latency figures in the table to calculate the I–II and II–III interwave duration figures in the table for the right side of the experimental subjects.

[b] $t = 2.511$; $p < 0.05$; df 1, 9.

[c] $t = 2.264$; $p < 0.05$; df 1, 9.

[d] $t = 2.738$; $p < 0.05$; df 1, 9.

[e] $t = 3.000$; $p < 0.05$; df 1, 8.

stimulation ($t = 2.511$; $df = 1,9$; $p < 0.05$) and for wave I on right ear stimulation ($t = 2.738$; $df = 1,9$; $p < 0.05$).

When one examines for intra-group right- vs. left-ear stimulation latency and interwave transmission time differences, none were found for the experimental subjects (Table 2). The controls, however, did show a longer wave II latency on the right ($t = 3.207$, $df = 1,9$; $p < 0.05$) and a longer I–II interwave transmission time on the right ($t = 3.972$, $df = 1,9$; $p > 0.01$) as compared to the left.

The ANOVA showed a significant interaction ($f = 5.82$; df 1,39; $p < 0.05$) between laterality and age for the amplitude of wave IV-V. Significance was also seen in the triple interaction (subject groups, laterality, and age) for the amplitude of wave III. t-Tests showed no significant differences between the groupings (experimental subjects vs. controls, younger vs. older, left vs. right) for any of the wave amplitudes. However, when the experimental subjects and the controls were combined into one group ($N = 20$), the amplitude of wave III for right ear stimulation was significantly greater than it was for left ($t = 2.35$; df 1,19; $p < 0.05$) (Table 3). The laterality differences in the amplitude of wave III were large for both the experimental subjects (0.08 μV) and for the controls (0.09 μV) and did not increase for the two groups combined (0.09 μV), but the larger N (group of 20) and resulting increase in the degrees of freedom from 9 to 19 resulted in the difference reaching significance.

Discussion

In this study the sample was small but the controls were paired for age and sex with the experimental subjects. All were medication free, and none were mentally retarded.

It would appear that the transmission time from onset of the sound to the superior olives as represented by wave III is longer in the children with evidence of central language disturbance compared to children without such evidence. All segments of the period from stimulation to wave III bilaterally with the exception of the I–II interwave duration on right ear stimulation are longer for the experimental subjects than for the controls. The differences reached significance for some segments and not for others. On the left, the experimental subjects showed a significantly longer wave III latency and a significantly longer I–III interwave duration. On the right, the wave III latency difference did not reach significance, but two of its segments, the wave I latency and the II–III interwave duration, were significantly longer in the experimental

TABLE 2

Latency and Interwave Durations (ms) Left Side vs. Right Side

	Latency						Interwave Transmission Time					
	Wave I		Wave II		Wave III		I–II		II–III		I–III	
	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right
Experimental subjects												
X	1.42	1.43	2.54	2.50	3.61	3.58	1.12	1.09	1.06	1.03	2.18	2.15
S.D.	0.15	0.08	0.15	0.08	0.14	0.19	0.08	0.09	0.11	0.07	0.17	0.16
Controls												
X	1.36	1.33	2.41	2.48[a]	3.40	3.43	1.04	1.16[b]	1.00	0.93	2.04	2.10[b]
S.D.	0.09	0.09	0.13	0.13	0.15	0.16	0.10	0.12	0.11	0.08	0.13	0.14

[a] $t = 3.207$; $p < 0.05$; df 1, 9.
[b] $t = 3.972$; $p < 0.01$; df 1, 9.

TABLE 3

Amplitude (μV) Left Side vs. Right Side

	Wave I		Wave II		Wave III		Wave IV-V	
	Left	Right	Left	Right	Left	Right	Left	Right
Experimental subjects								
X	0.48	0.51	0.19	0.17	0.53	0.61	0.92	0.90
S.D.	0.02	0.01	0.01	0.01	0.03	0.03	0.03	0.03
Controls								
X	0.52	0.48	0.17	0.20	0.53	0.62	0.73	0.76
S.D.	0.03	0.02	0.01	0.01	0.02	0.02	0.02	0.02
Combined groups								
X	0.50	0.50	0.17	0.19	0.53	0.61[a]	0.83	0.83
S.D.	0.02	0.02	0.01	0.01	0.03	0.03	0.03	0.03

[a] $t = 2.353$; $p < 0.05$; df 1, 19

subjects than in the controls. This suggests that there is definite slowing of auditory transmission bilaterally prior to the impulse reaching the olivary nuclei in this group of children with language disturbance as compared to a group of children who did not have evidence of language disturbance.

Rosenblum et al. (1980) found significantly increased latencies for waves III and IV and for the transmission time from the positive peak of wave I to the negative peak of wave IV in their group of autistic children.

Fein et al. (1981) found significantly increased interwave durations bilaterally between waves I and V and on the left between waves III and V in their autistic children as compared to normal children. They found that these abnormalities correlated with aloofness and unusually long attention span but not with language disturbance. They had rated each autistic child on a scale of 1 to 9 for language expression and for language comprehension. Our findings of increased latencies prior to wave III as compared to theirs of increased latencies extending to wave IV (Rosenblum et al., 1980) and to wave V (Fein et al., 1981) may indicate that language is more likely to be affected by disturbances lower in the auditory brainstem pathways as compared to symptoms indicative of disturbances in social relatedness. Sohmer and Student's (1978) study of autistic, minimal brain dysfunctioned, and mentally retarded children also showed increased latencies extending to wave V. Many of those children had language disturbances. Here too it may be that the language disturbances relate to the longer latencies prior to wave III.

Of interest is Salamy and Mendelson's (1980) report comparing the pattern of BSAER in infants with perinatal complications to normal infants during the first year of life. They found slowing of waves I, III, and V in the high risk infants but convergence of the latencies of waves I and V during the first 5 months. Wave III latency, which had been closely parallel between the two groups, began to diverge in the latter part of the first year of life as the transmission time began to decrease more rapidly in the normals than in the high risk infants. They felt this might well represent measurable differences in brain stem transmission that would continue and might underlie or be a part of the problem seen later in children manifesting cognitive impairments and neurological soft signs. The work of Fein et al. (1981), Rosenblum et al (1980), and Sohmer and Student (1978) and our findings would appear to support this belief.

The controls also showed some laterality differences in the rate of transmission of the impulse from onset of the sound to the cochlear nuclei as represented by wave II, in that the wave II latency was significantly longer on the right ear stimulation. This increased duration was primarily in the I–II interwave duration. We also found a laterality difference in the amplitude of wave III; it was larger on the left for both our normal and experimental subjects, the difference reaching significance when both groups were combined. This is the opposite of what we would have expected if, as we had believed, the nerve transmission is symmetrical. Our findings would suggest there may be a normal assymetry in the brain stem transmission of sound, but at this point whether or not this is a real finding or an artifact is unclear.

The children in this study were paired for age and sex and were medication free so that any effects due to these factors would not be expected to influence the results. The study was not controlled for time of day; i.e., some subjects were tested in the morning, others in mid-afternoon, thus temperature variations related to circadian rhythm as demonstrated by Marshall and Donchin (1981) might have introduced distortions into our findings. The experimental subjects were screened audiometrically an average of 8 months before the BSAER testing. There is the possibility that some of these children could have developed a hearing loss in the interval and the findings are secondary to undetected hearing loss. This conceivably could explain the findings on right ear stimulation where wave I latency was significantly longer in the experimental subjects than in the controls. It could not explain the difference on left ear stimulation, however, where the wave I latency difference between the two groups was not significant. The significant slowing came after wave I. The high correlation (0.914) between the two raters for wave I left ear stimulation compared to the lower (but still significant) correlation (0.636) between the raters for wave I right ear stimulation would indicate the left ear stimulation findings may be more reliable, and that the slowing is not due to hearing loss. Also, in the interval CNS maturation could have decreased the transmission disturbances so that we would have been less likely to obtain positive findings. The data do indicate the possibility of transmission deviations at the brainstem level in children with language disturbances. There is indication that a number of children with various emotional and intellectual disturbances have language disturbances. Many of these children also show indications of transmission deviations on BSAER studies. One can question the degree to which these brainstem deviations actually do represent a physiological brainstem abnormality and how much they represent recording and statistical analysis artifact. However, they have been found in a number of groups of disturbed children, many of whom have disturbances in language. We have now demonstrated

them in a group of children with normal hearing who were specifically chosen because they had language disturbance. We would speculate that at least a portion of the slowed brainstem transmission findings in many children with diagnoses of retardation, autism, and minimal brain dysfunction may relate to language disturbance.

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